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Aims: This study aims to systematically summarize the frequency of ATE in patients with cancer reported through observational studies.

Methods: A comprehensive literature review of MEDLINE, Embase, CENTRAL, and Web of Science from inception to Nov 28, 2018 was conducted. Two independent reviewers screened for eligibility using pre-established criteria; studies comparing the frequency of ATE between populations with cancer and healthy controls were included while studies examining the frequency of ATE in the context of therapies (e.g., chemotherapy, radiotherapy) were excluded. Data corresponding to the follow-up times closest to initial diagnosis and closest to 1 year were extracted.

Results: 10 retrospective cohort studies involving 1,228,897 patients were eligible for inclusion, summarized in Table 1 and 2. Eight studies found increased occurrence of ATE in populations with malignancies. At the time-point closest to diagnosis, all analyzed cancers except prostate cancer increased ATE. Studies unanimously agreed that lung, bladder, colorectal, pancreatic, and non-Hodgkin lymphoma increased ATE as well as myocardial infarction and ischemic stroke independently. This risk diminished at the time-point closest to 1 year with the exception of lung and pancreatic cancer. High heterogeneity within and between studies due to the different databases used, unknown interventions provided, and reported measures precluded meta-analysis.

Conclusions: Patients with cancer appear to have an increased risk of developing ATE, with the highest risk immediately following the time of diagnosis and in lung and pancreatic cancers. High heterogeneity suggests the need for future high-quality, prospective studies that record comprehensive patient characteristics and interventions provided to patients.

PB1160 | Combination of High Levels of Fibrinogen and High Sensitivity C-reactive Protein Enhances Risk of Acute Coronary Syndrome

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Background: Increased levels of fibrinogen and high sensitivity C-reactive protein (hs-CRP) in unstable coronary artery disease (CAD) patients are correlated with the occurrence of cardiovascular event.

Aims: To investigate whether combination of these two parameters could enhance the prediction of acute coronary syndrome (ACS) in patients with CAD.

Methods: A case-control study included 70 CAD patients presenting a symptom of ACS and 55 controls who underwent cardiac catheterization for atypical chest pain and had no significant coronary

stenosis based on coronary angiography results. Levels of plasma fibrinogen were determined by using the automated coagulometer based on a modified Clauss method whereas hs-CRP levels were measured by automated analysis based on nephelometric principle. Association of fibrinogen and hs-CRP with ACS risk was further investigated by using binary regression analysis.

Results: Odds ratios after adjustment for age, gender and diabetes mellitus showed that subjects with higher levels of fibrinogen (fibrinogen ≥ 328.5 mg/dL) [adjusted OR (95%CI) = 2.7 (1.2, 6.1)] and hs-CRP (hs-CRP ≥ 2.9 mg/L) [adjusted OR (95%CI) = 7.6 (3.1, 18.9)] were associated with ACS. Moreover, combined effect of these two parameters on ACS risk revealed that subjects with high hs-CRP levels but low concentration of fibrinogen were significantly associated with ACS [adjusted OR (95%CI) = 4.8 (1.3, 17.9)] whereas a significant association was not found in the individuals with high fibrinogen but low hs-CRP. Interestingly, a combination of high fibrinogen levels and high hs-CRP levels was associated with an increased ACS risk [adjusted OR (95%CI) = 9.4 (3.1, 28.7)].

Conclusions: This study suggested that a combination of high plasma levels of fibrinogen and hs-CRP enhanced risk of acute coronary syndrome. Thus, both biomarkers may be used as a co-predictor for ACS in patients with CAD.

PB1163 | Demographic, Clinical and Hematological Predictors of Carotid Atherosclerotic Plaque Histology

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Background: Carotid atherosclerotic disease (CAD) is the third leading cause of disability and mortality in Western countries, so that cost-effective strategies for preventing and early detecting severe disease are highly advisable.

Aims: This retrospective observational study was aimed to identify hematological predictors of histological heterogeneity of carotid atherosclerotic plaques (CAPs).

Methods: CAP histology was assessed in 135 patients (mean age, 74 \pm 8 years; 44 women and 91 men; 31 with symptomatic CAD and 104 without), using a semiquantitative scale. Plaque specimens were embedded in paraffin, stained with hematoxylin and eosin, and analyzed at three levels by skilled histopathologist. The evaluated CAP features included the presence of intraplaque haemorrhage, superficial thrombosis, lipid core, fibrosis, foam cells, neovascularization, calcification, inflammatory infiltrate (plaque or fibrous cap) and fissuration/cap rupture. Results of histopathological assessment were compared between symptomatic and asymptomatic subjects, and then correlated (Spearman's correlation)

with clinical history and hematological parameters, including white blood cell (WBC) count, red blood cell (RBC) count, platelet (PLT) count, mean corpuscular volume (MCV) and RBC distribution width (RDW). The study was cleared by the local Institutional Review Board.

Results: The mean values of all demographic, clinical and haematological parameters did not differ between patients with or without symptomatic CAD. In univariate analysis, intraplaque hemorrhage was associated with male sex ($r=0.18$; $p=0.032$), superficial thrombosis with low hemoglobin ($r=-0.18$; $p=0.033$), fibrosis with enhanced RDW ($r=0.24$; $p=0.005$), presence of foam cells with high WBC count ($r=0.22$; $p=0.001$), neovascularisation with high WBC count ($r=0.17$; $p=0.048$), whilst the presence of inflammatory infiltrate was also associated with high WBC count ($r=0.17$; $p=0.043$).

Conclusions: The results of this retrospective observational study confirm that some traditional and inexpensive hematological parameters such as WBC count, hemoglobin and RDW may help identifying patients with more severe forms of CAD.

PB1164 | Endocan and Homocysteine Levels in Brazilian Renal Transplant Recipients

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Background: Although the success of Kidney Transplantation, the recipients still showed a high risk of adverse outcomes such as endothelial dysfunction and cardiovascular events. Endocan and Homocysteine are new markers for these events, could be potential models to evaluated post-transplant. Endocan plays a role in cell adhesion and inflammatory disorders. Homocysteine exerts its pathogenic action on the main processes involved in progression of vascular damage.

Aims: Our aim was to investigate the relationship between Endocan and Homocysteine levels with evolution of allograft in renal transplant recipients (RTR).

Methods: In cross-sectional study, a total of 100 RTR were allocated in groups according to creatinine levels (C1 \leq 1.5 and C2>1.5mg/dL); eGFR (R1 \leq 60; R2>60mL/min/1.73m²); time post-transplantation (T1 \leq 60; T2=61 to 119; T3 \geq 120 months) and Donor type (Living or Deceased). Endocan plasma levels and Serum Homocysteine levels were measured by ELISA methods. All data are presented as median and interquartile range (Mann-Whitney U-test or Kruskal-Wallis followed by Dunn's). $P < 0.05$ was significant.

Results: Higher levels of endocan and homocysteine were found in RTR with worst renal filtration function (C2 and R1 groups) in relation to the others (Figure 1A, 1B, 1C and 1D). There was no

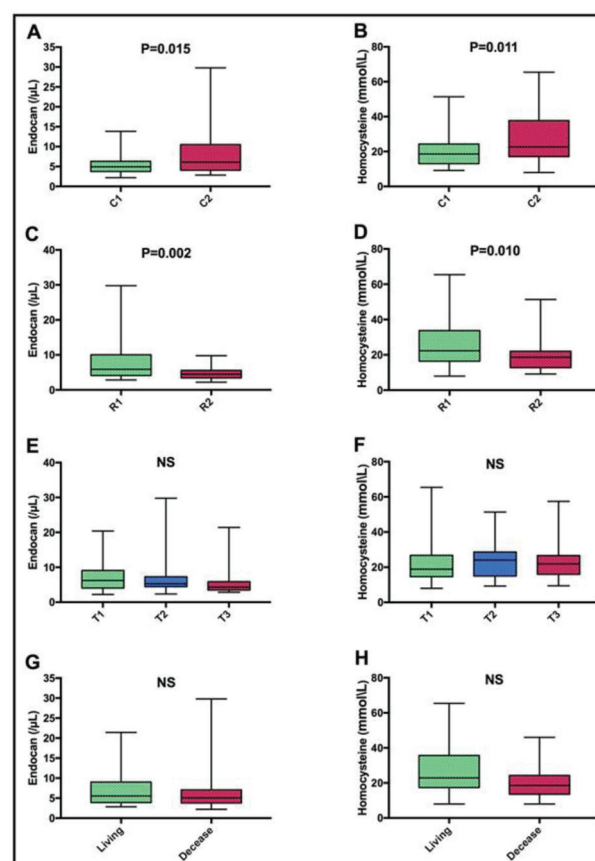


FIGURE 1 Endocan and Homocysteine in RTR. A-B:Creatinine; C-D: eGFR MDRD; E-F: Time post-Tx; G-H: Donor

difference among time post-transplantation and donor type groups (Figure 1E, F, G and H).

Conclusions: Endocan and homocysteine levels were promising indicators of cardiovascular and cardiorenal risk in RTR, with association to renal function. Donor type and time post-transplantation showed no influence in mechanism of rising levels of endocan and homocysteine. However, further studies are needed to investigate the role of these biomarkers as a tool after transplantation.

Support: Capes, CNPq and FAPEMIG.

PB1166 | An Intracardiac Thrombus Associated with Antithrombin Deficiency with a Novel Mutation c.1063T>G in SERPINC1

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Background: Antithrombin(AT) is one of the most important inhibitors of blood coagulation. The most important complication of AT